Isolation of a Dengue Type 1 Virus from a Soldier in West Africa (Côte d'Ivoire)

To the Editor: In Africa, recent reports of epidemic or endemic dengue activity usually come from the eastern part of the continent (1), and the serotype most frequently identified is dengue 2.

We report the isolation of a dengue 1 virus strain from the blood of a young soldier living in Abidjan, the capital of Côte d'Ivoire. This 20-year-old man arrived from France on October 19, 1998. On December 28, 1998, he consulted a physician of his regiment because of headache, diarrhea, and fever (40°C). The results of the laboratory investigations were as follows: platelets (193 10^9 /L), leukocytes (2.2$10^9$ /L); no malaria was found. He was hospitalized for possible arboviral infection, and treatment with paracetamol was prescribed. On December 29, a blood sample was collected; serum and buffy coat were frozen at -20°C for further examination at the Institute of Tropical Medicine of the Military Health Services. On day 3, the patient's temperature dropped to 38°C, then rose to 39.5°C on day 5. All symptoms were resolved on day 6.

On February 15, 1999, the frozen blood was defrosted and the lysed buffy coat was immediately cocultured with C6/36 cells. On day 6, a blind passage was made on the same cells and on Vero. On day 12, no cytopathic effect was observed, but a dengue 1 virus was identified by indirect fluorescent antibody assay (2) with type-specific monoclonal antibodies. This diagnosis was confirmed by reverse transcription-polymerase chain reaction, with a technique slightly modified from Lanciotti (3).

On the first blood specimen, the serologic immunoglobulin (Ig)M assays (M antibody capture enzyme-linked immunosorbent assay [ELISA]) with our antigen screening panel were negative (dengue, West Nile, Chikungunya, Rift Valley fever). The IgG assay (ELISA) against dengue antigen was also negative.

The patient returned to France in February 1999. A second blood sample was collected and tested on April 7, 1999, 3 months after the illness. The serologic assays were positive against the dengue antigens at the following dilutions of the serum: IgM: dengue 1: 1/120,000; dengue 2: 1/40,000; dengue 3: 1/12,000; dengue 4: 1/25,000. IgG: dengue 1: 1/75,000; dengue 2: 1/90,000; dengue 3: 1/60,000; dengue 4: 1/120,000. This seroconversion allowed us to confirm infection of this patient by a dengue virus.

The lack of similar reported cases or epidemics among the local and expatriate populations of Abidjan may indicate poor transmission, recent introduction of the strain, or low virulence of the virus, as previously hypothesized for dengue in West Africa (4). However, the serologic status of the human population needs to be further investigated. Characterization of the isolated viral strain would be of interest for dengue epidemiology. Complete sequencing of the viral RNA is in progress in our laboratory.

Human infection with dengue virus has been rarely reported and studied in West Africa, and the epidemiology of serotype 1 is poorly documented. During the past 35 years, the Pasteur Institute of Dakar confirmed three dengue 1 strains (two from humans in Senegal; one from mosquitoes in Côte d'Ivoire), while during the same period, more than 300 dengue 2 strains were studied, most from mosquitoes (5). In the past 10 years, medical and entomologic surveys showed circulation of dengue 2 virus in Senegal (one isolate in the blood of a French soldier) (6). However, during the 1970s, Nigerian virologists demonstrated circulation of dengue 1 and 2 in their country: more than 50% of the adults living in the savannah had neutralizing antibodies (7). Of 148 blood samples of febrile patients, three viral strains (yellow fever, dengue 1, and Zika) were isolated, all from children (8).

In Africa, outside the epidemics of yellow fever, it is difficult to isolate arboviruses from adult humans. Isolation is often more successful from children or naive expatriates. Accordingly, soldiers participating in international operations constitute a very exposed population. During recent operations in Somalia (1992-93), dengue fever was an important cause of febrile illness among U.S. troops (9). Thirty-nine dengue 2 and three dengue 3 strains were isolated from 96 collected sera.

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Carbapenem-Hydrolyzing Metallo-ß-Lactamase from a Nosocomial Isolate of *Pseudomonas aeruginosa* in France

To the Editor: The carbapenems (meropenem and imipenem), the ß-lactams with the broadest spectrum, are stable to most ß-lactamases (1). Therefore, they are often used as antibiotics of last resort for treating nosocomial infections due to gram-negative bacteria resistant to other ß-lactams. Resistance to carbapenems and susceptibility to other ß-lactams in *Pseudomonas aeruginosa* is common as a result of reduced drug accumulation or increased expression of pump efflux (1).

Several extended-spectrum ß-lactamases have been reported in *P. aeruginosa*, but only two, IMP-1 and VIM-1, possess an extended hydrolysis profile that includes carbapenems (2-5). The chromosome-borne and plasmid-mediated carbapenem-hydrolyzing ß-lactamase, IMP-1, has been described in several gram-negative rods, including *P. aeruginosa*, *P. cepacia, Alcaligenes xylosoxydans,* and *Enterobacteriaceae* isolates in Japan (4,6). Recently, a chromosome-borne carbapenem-hydrolyzing ß-lactamase, VIM-1, was reported from a clinical isolate of *P. aeruginosa* in Italy (5), and uncharacterized carbapenem-hydrolyzing ß-lactamases have been reported in the United Kingdom and Portugal (7,8). The weakly related IMP-1 and VIM-1 (31.4% amino acid identity) are both zinc-dependent (metallo-enzymes) and confer resistance to all ß-lactams except monobactams (3,5).

In 1996, a 39-year-old French woman was hospitalized in Marseille for chronic myelogenous leukemia, pancytopenia, and allogeneic bone marrow transplantation. After a 15-day stay in the transplantation unit, fever developed and imipenem and amikacin were administered. Despite this treatment, the patient died of septic shock syndrome 5 days later. Three-day-old blood cultures grew a carbapenem-resistant *P. aeruginosa* isolate. This *P. aeruginosa* COL-1 isolate was resistant to most ß-lactams, including piperacillin/tazobactam, imipenem, meropenem, ceftazidime, cefepime (minimum inhibitory concentrations [MICs] of 128, 32, 16, 64, 32 mg/L, respectively), amikacin, tobramycin, gentamicin, netilmicin, and ciprofloxacin; however, the isolate was susceptible to aztreonam (MIC determination, genetic techniques and ß-lactamase assays are described elsewhere [9]). A sonicate of crude extract of *P. aeruginosa* COL-1 culture showed strong imipenem and meropenem hydrolysis activity (0.7 mU/mg and 1.9 mU/mg; reference *P. aeruginosa* strain <0.05 mU/mg) by UV spectrophotometry with 0.1 mM of substrate, after incubation in 50 mM phosphate buffer at 30°C. This activity was lost when the enzyme extract was preincubated with 10 mM of edetic acid and was partially restored

by addition of 1 mM $ZnCl_2$, indicating the presence of a metallo-carbapenem hydrolyzing ß-lactamase. Isoelectric focusing revealed two ß-lactamase bands of pI 5.6 and 9. Only the pI 5.6 ß-lactamase band was inhibited if the gel was overlaid with edetic acid before nitrocefin was added as the indicator substrate; the other pI 9 ß-lactamase likely corresponded to a naturally occurring AmpC cephalosporinase. This pI 5.6 value differed from the pI values of the carbapenem-hydrolyzing ß-lactamase previously reported in *P. aeruginosa* (3-5,7,8). Polymerase chain reaction amplification experiments were negative when internal primers were used for the only sequenced carbapenem-hydrolyzing ß-lactamase genes from *P. aeruginosa* encoding IMP-1 and VIM-1 and genomic DNA of *P. aeruginosa* COL-1. Transfer of the carbapenem resistance marker by conjugation to laboratory strains of *P. aeruginosa* or *Escherichia coli* was unsuccessful (9), but transformation by electroporation of a putative plasmid extract from *P. aeruginosa* COL-1 in *E. coli*, followed by selection onto amoxicillin-containing agar plates (9), gave a ca. 45-kb plasmid that produced the carbapenem-hydrolyzing ß-lactamase with a pI value of 5.6. Thus, the carbapenem-hydrolyzing ß-lactamase gene was plasmid-borne.

This case indicates the presence of a novel carbapenem-hydrolyzing ß-lactamase in *P. aeruginosa* in Europe, the first in France; its spread in gram-negative rods, as reported for IMP-1 in Japan, is of concern because, as seen in this case, routine laboratory detection is difficult and therapeutic options are extremely limited.

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Population-Based Study of Invasive *Kingella kingae* Infections

To the Editor: For most of the 3 decades since the first description of *Kingella kingae*, this gram-negative bacillus was considered a rare cause of human disease (1). Since the late 1980s, however, reports of infections by the organism in young children have increased in the United States, Western Europe, and Israel (2-6). The rapid emergence of *K. kingae* as an important cause of pediatric disease does not necessarily imply that the organism is truly a new pathogen. Better isolation techniques and awareness of the bacterium by microbiology laboratories may contribute to the apparent increase (4). Recent studies have demonstrated that primary isolation of *K. kingae* can be substantially improved by injection of synovial fluid and bone exudates into aerobic blood-culture bottles (4). Synovial fluid may inhibit the growth of *K. kingae,* and injection of the clinical specimen

into a 50-mL volume of broth reduces the concentration of inhibitory factors, facilitating isolation of the organism (4).

In 1993, we reported results of routine use of blood-culture bottles for processing cultures of exudates (7) at the Soroka University Medical Center, Beer-Sheva, Israel. From 1988 to 1992, 25 children with invasive *K. kingae* infections, defined as isolation of the organism from blood or normally sterile body fluids, were identified in southern Israel. From 1994 to 1998, 33 additional patients, including 32 children and a 21-year-old adult, were detected in the same area. Twenty-four (63.6%) of the 33 patients were male. Eight (24.2%) cases were diagnosed between January and June and 25 (75.8%) between July and December. Median age of children was 13 months (mean ± SD: 15.0 ± 7.6 months; range 6 to 37 months).

The fact that all children in southern Israel are born and receive inpatient medical services at the Soroka University Medical Center allowed us to calculate the incidence of invasive pediatric *K. kingae* infections in this population. During the 6-year period, the average annual number of births was 10,860. The annual incidence of invasive *K. kingae* infections during the same period was 11.9 per 100,000 in children ≤ 48 months of age, 19.2 per 100,000 in children <24 months of age, and 20.0 per 100,000 in infants <12 months of age.

When medical attention was sought, patients had been ill for a median of 3 days. Symptoms of upper respiratory tract infection were recorded in 12 (36.4%) children, stomatitis in 8 (24.2%), and diarrhea in 4 (12.1%). Occult bacteremia (positive blood culture with no obvious focal infection) was diagnosed in 16 children. In 15 children, *K. kingae* had invaded the bones. Septic arthritis was diagnosed in 11 children, involving the ankle in 4; the knee or wrist in 2 patients each; and the hip, shoulder, or elbow in one patient each. Osteomyelitis was diagnosed in two patients, affecting the femur in one and the tibia in the other. In two additional patients, both with fever and bacteremia, the location of the skeletal infection could not be determined. One limped and had tenderness over the femur, but X-rays and a $Technecium_{99}$-labeled bone scan showed no abnormalities. The other had pain in the heel but no fluid could be aspirated. Bacteremic tracheobronchitis occurred in one child, and endocarditis of the mitral valve was diagnosed in a 21-year-old woman who was receiving immunosuppressive therapy for systemic lupus erythematosus. All 33 patients were treated with ß-lactam drugs and recovered.

Injecting synovial fluid specimens into blood-culture bottles permitted the diagnosis of *K. kingae* in these patients and showed that this organism may be a common cause of invasive pediatric infections. The age distribution of the patients demonstrates that *K. kingae* is a pathogen of young children, especially those between the ages of 6 months and 2 years, among whom the incidence of invasive disease has remained stable since 1988. This age distribution of *K. kingae* infections parallels that for respiratory carriage of the organism. In a surveillance study among 48 children ages 6 to 42 months attending a day-care center in Israel, *K. kingae* was isolated from 109 (17.5%) of 624 throat cultures, and 34 children (70.8%) carried the organism at least once during an 11-month period (8). However, the organism was not detected in healthy infants ages 2 to 4 months attending a well-baby care clinic, which indicates some immunity to colonization and infection by *K. kingae* during the first months of life (8).

When the 1988 to 1993 surveillance data are added to those collected from 1994 to 1998, *K. kingae* infections show a significant seasonal pattern; 44 (75.9%) of 58 cases were diagnosed in the second half of the year ($p = 0.007$). This increase in *K. kingae* infections in winter has also been described in other respiratory pathogens. This finding, as well as the frequent detection of respiratory symptoms in children with invasive *K. kingae* infections, suggests that seasonal viral infections may facilitate the spread of *K. kingae* from the throat, to the bloodstream and bones. In a prospective study, *K. kingae* bacteremia was documented in 4 (13.7%) of 29 young children with culture-proven herpetic gingivostomatitis, confirming the role played by viral infections in the pathogenesis of infections caused by the organism (9).

With few exceptions, isolates of *K. kingae* remain susceptible to antibiotic drugs (10). Our results demonstrate that the prognosis of invasive *K. kingae* infections is generally good and patients respond promptly to appropriate antimicrobial therapy.

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Involving Ornithologists in the Surveillance of Vancomycin-Resistant Enterococci

To the Editor: Because migratory birds cross national or intercontinental borders, they are possible long-range vectors for human pathogens such as viruses, *Borrelia burgdorferi* sensu lato, and enteropathogenic bacteria with antibiotic resistance or virulence factors (1,2). Enterococci are ubiquitous in humans and animals and have a propensity for uptake and transfer of glycopeptide antibiotic resistance (3); therefore, the emergence of glycopeptide-resistant enterococci (GRE) in humans is a public health concern. Low-level vancomycin resistance (genotype *vanC-1-3*) is intrinsic in enterococcal species (e.g., *Enterococcus gallinarum, E. flavescens,* and *E. casseliflavus*) that may normally occur in the intestinal flora of some birds. However, the finding of high levels of GRE in wild birds suggests acquisition from an environmental source.

In March 1998, we obtained fecal samples while banding 318 northbound migrating gulls in Malmö, southern Sweden. Using a selective culture procedure with enrichment broth (bile esculin azide broth, Acumedia, LabFab, Ljusne, Sweden) containing vancomycin (8 µg/ml) and aztreonam (60 µg/ml), we isolated vancomycin-resistant *E. faecalis* from a black-headed gull (*Larus ridibundus*). High-level glycopeptide resistance (>256 µg/ml) was demonstrated by E-test (AB Biodisc, Solna, Sweden), and a *van*A genotype was found by polymerase chain reaction amplification (4). This survey protocol can also be used to detect medium to low levels of glycopeptide resistance. Using the same procedure in a study of 230 sub-Antarctic birds on Bird Island, South Georgia, in 1996, we found four GRE isolates with *vanC1* genotype (MIC 3-8 µg/ml).

Many species of gulls have moved into urban areas, where they commonly feed on human trash and deposit feces. The black-headed gull with GRE described above was banded as a fledgling in Malmö in 1995. Birds of this population spend the winter mainly in Western Europe (5), where they forage at garbage dumps, sewage outlets, and agricultural areas. This bird may have acquired GRE in such an area. *Van*A genotype *E. faecium* and *E. faecalis* have been found in poultry and pigs in the Netherlands and Denmark, where the vancomycin analog avoparcin has been used as a growth promoter (6). Manure from such farms may be a GRE source accessible to wild birds.

We have previously reported the introduction into Sweden of multidrug-resistant *Salmonella* Typhimurium by migratory birds (7). The present report further emphasizes the possibility of migratory birds as long-range vectors of bacteria potentially associated with human disease. The risk to humans for GRE from migratory birds may seem insignificant compared with such risk from hospitalization or from eating meat products from GRE-colonized

animals. However, if the frequency of birds carrying high-level GRE increases and if amplification in a secondary reservoir or spread through polluted water takes place, spread by migratory birds may become a problem. Bacteriologic surveys of birds may provide vital information for assessing the environmental dispersion of GRE from farms and hospitals. In combination with data about migration patterns and reports of banding recoveries from ornithologists, the potential sources of GRE might be deduced.

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